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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/618,856	07/15/2003	Beka Solomon	SOLOMON=2A.1	6926
1444	7590	12/29/2005	EXAMINER	
BROWDY AND NEIMARK, P.L.L.C.			TURNER, SHARON L	
624 NINTH STREET, NW				
SUITE 300			ART UNIT	PAPER NUMBER
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DATE MAILED: 12/29/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

16

Office Action Summary	Application No.	Applicant(s)
	10/618,856	SOLOMON ET AL.
	Examiner	Art Unit
	Sharon L. Turner	1649

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --
Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1) Responsive to communication(s) filed on 19 October 2005.
 2a) This action is FINAL. 2b) This action is non-final.
 3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4) Claim(s) 1-12 is/are pending in the application.
 4a) Of the above claim(s) 9 and 11 is/are withdrawn from consideration.
 5) Claim(s) _____ is/are allowed.
 6) Claim(s) 1-8, 10 and 12 is/are rejected.
 7) Claim(s) _____ is/are objected to.
 8) Claim(s) 1-12 are subject to restriction and/or election requirement.

Application Papers

9) The specification is objected to by the Examiner.
 10) The drawing(s) filed on 15 July 2003 is/are: a) accepted or b) objected to by the Examiner.
 Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
 Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
 11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
 a) All b) Some * c) None of:
 1. Certified copies of the priority documents have been received.
 2. Certified copies of the priority documents have been received in Application No. _____.
 3. Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) Notice of References Cited (PTO-892) 4) Interview Summary (PTO-413)
 2) Notice of Draftsperson's Patent Drawing Review (PTO-948) Paper No(s)/Mail Date. _____.
 3) Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)
 Paper No(s)/Mail Date _____. 5) Notice of Informal Patent Application (PTO-152)
 6) Other: _____.

Detailed Action

1. This Office Action is supplemental to the action of 12-9-05 and is to correct the inadvertent error with respect to the previous time period for response. It resets the time period for response to 3 months.
2. The amendment filed 10-19-05 has been entered into the record and has been fully considered.
3. Claims 1-12 are pending and under examination. Claims 13-17 have been canceled.

Election/Restrictions

4. Applicant's election of Group I, claims 1-12, in the reply, filed 3-8-05 is acknowledged. Because Applicant did not distinctly and specifically point out the supposed errors in the restriction requirement, the election has been treated as an election without traverse (MPEP § 818.03(a)).

Applicant's election of species of SEQ ID NO:1 (EFRH) in the reply filed on 10-19-05 is acknowledged. Because applicant did not distinctly and specifically point out the supposed errors in the restriction requirement, the election has been treated as an election without traverse (MPEP § 818.03(a)). Applicants state that all claims read on the elected species as SEQ ID NO:1 is shared amongst all sequence members. However, a search for SEQ ID NO:1 is not co-extensive to all other members. Further SEQ iD NO:1 is anticipated by the prior art as noted below. Accordingly, claim 8 is presently non-linking with respect to SEQ ID NO:1 and the other sequences denoted in claim 9.

Claim 9 and 11 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected species, there being no allowable generic or linking claim.

Priority

5. Applicant's claim for domestic priority under 35 U.S.C. 119(e) is acknowledged. However, the provisional application upon which priority is claimed fails to provide adequate support under 35 U.S.C. 112 for the limitations of claims 5, and 7. Claim 11 depends from claim 9 and thus also lacks specific support from the provisional. Accordingly the effective filing date for the limitations recited in these claims is the filing date of 7-15-03. Traversal should indicate where specific support for the noted limitations may be found within the provisional application.

Sequence Compliance, Specification and Drawings

6. The disclosure is objected to because of the following informalities: The specification and figures fail to comply fully with the sequence rules as Figure 25 appears to reference amino acid sequences which are not referred to by their appropriate SEQ ID NO: either within the figure or within the brief description of the drawings.

Appropriate correction is required.

Double Patenting Rejections

7. The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. See *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA

1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and, *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.130(b).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

8. Claims 1-8, 10, and 12 are provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-20, especially claims 11-13 and 17-20 of copending Application No. 11/073,526. Although the conflicting claims are not identical, they are not patentably distinct from each other because the '526 claims stipulate a species of the instant invention thereby fairly anticipating instant generic claims. In particular, the instant application claims a method of inhibiting aggregation of β -amyloid in a subject or disaggregating aggregated β -amyloid in a subject comprising administering to a subject in need thereof an effective amount of a filamentous bacteriophage which displays an epitope of β -amyloid so as to elicit antibodies against said epitope in said subject, wherein said antibodies inhibit aggregation of said β -amyloid in said subject and/or cause disaggregation of said β -amyloid aggregate in said subject. The application 11/073,526 claims a method of treating Alzheimer's disease by introducing a pharmacological composition in a subject in need thereof to inhibiting aggregation of β -amyloid in a subject and treat the disease. The subject pharmaceutical compositions are directed to the same, a phage virus particle displaying a polypeptide that comprises at least one epitope of beta amyloid (claim 1 of '526). In the '526 Application, claim 13 recites the virus is a bacteriophage,

and claim 17 recites to inhibit aggregation of beta-amyloid as in claim 1 of the instant application. The instant application does not teach administering the bacteriophage as a composition to treat Alzheimer's disease. However, the treatment of Alzheimer's disease is a species member of diseases recognized as requiring inhibition of aggregation of β -amyloid peptide. Since both applications teach inhibiting aggregation or removal of amyloid, the patenting of the co-pending claims would render obvious the instant Application directed to the generic mechanism of action or function of the Alzheimer's treatment. This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

Claims 1-8, 10 and 12 are rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-19 of U.S. Patent No. 6919075. Although the conflicting claims are not identical, they are not patentably distinct from each other because the co-pending claims are drawn to methods comprising administration of the same product composition as similarly noted above. In particular the compositions overlap as comprised of bacteriophage display of beta amyloid epitopes including of SEQ ID NO:1 for treatment of Alzheimer's disease via inhibition of aggregation of beta-amyloid in the host.

Claim Rejections - 35 USC § 102

9. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

- (a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

(e) the invention was described in (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent or (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effects for purposes of this subsection of an application filed in the United States only if the international application designated the United States and was published under Article 21(2) of such treaty in the English language.

10. Claims 1, 8 and 12 are rejected under 35 U.S.C. 102(a) as being anticipated by Schenk et al. WO 99/27944, 10 June 1999 (IDS AE, 7-15-03).

11. Claims 1, 8 and 12 are also rejected under 35 U.S.C. 102(e) as being anticipated by, Schenk 6,787,139 Sept 7, 2004.

The aforementioned WO 99/27944 and 6,787,139 references are cumulative.

The patent extends to priority dates of 60/067,740 (12-02-97) and 60/067,740 (04-07-98). The citations noted below are in reference to the WO 99/27944 publication.

However the teachings are identical and the reasoning is therefore noted to be the same for both references.

Schenk et al. teach administration of β -amyloid immunogens (pg 3) to a patient in order to generate antibodies to prevent formation of amyloid plaques or dissolve existing plaques. The epitopes administered are inclusive of multiple species within or full length β -amyloid of recognized species 1-40, 1-42 and 1-43 and accordingly comprise the noted epitopes of SEQ ID NO:1, Such plaques are of aggregated Beta-amyloid in a subject, particularly of Alzheimer's disease patient. The plaques are areas of disorganized neuropil with extracellular amyloid deposits at the center visible by microscopic analysis of sections of brain tissue (Background). The reference teaches administration including intranasally, as in claim 12 (pg 4, line 17), of an agent, which includes immunogens, antibodies, active fragments, or derivatives of the antibody (pg 3,

Art Unit: 1649

line 6-8), effective to induce an immunogenic response against aggregated A β in a patient as in claim 1 (pg 2, line 34-36; pg 3, lines 18-20). The reference teaches A β 39, 40, 41, 42, or 43 (pg 3, line 28 and Summary); thus, comprising the relevant sequences as claimed in claim 8 and 9 (SEQ ID NO:3, A β 1-43). The reference teaches analysis of an effective amount via measurement of antibody titers and notes amounts inclusive of 1.0 μ g or 10 μ g or 50 μ g or 100 μ g (pg 3, line 25-7) effective to elicit antibodies and to effect removal or inhibition of plaque production/deposition. Schenk teaches mice, which developed a high antibody titer, including titers greater than 1/10,000 (see Figure 1). The highest antibody titers were generated with Freund's adjuvant with a peak GMT of about 87,000 as determined by ELISA. Schenk discloses if monitoring indicates a reduction of the immune response over time, the patient can be given one or more further doses of the agent (pg 4). Schenk discloses administration under conditions to generate a beneficial immune response (pg 2, line 35). Schenk et al. also teach the agent administered may be displayed via nucleic acid production for presentation within the host and expressed upon the surface of a virus or bacteria, specifically via phage (bacteriophage) display methods (pg 3, line 28; pg 16, and 17, line 10-11) with reference noted therein to the art recognized teachings of Devlin, WO91/18980 as in claim 1 and that peptide libraries can be generated by phage display methods (pg 16-7). Thus, the reference teachings anticipate claims 1, 8 and 12.

Claim Rejections - 35 USC § 103

12. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

13. This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

14. Claims 1-12 are rejected under 35 U.S.C. 103(a) as being unpatentable over either Schenk et al. WO 99/27944, 10 June 1999, or Schenk 6,787,139 Sept 7, 2004 (cumulative to WO 99/27944 priority extending to 60/067,740 (12-02-97) and 60/067,740 (04-07-98)), each alternatively in view of Devlin, WO91/18980, 12 December 1991, Willis A, Gene 128(1):79-83, 1993 (IDS 7-15-03, CC), Solomon et al., 1997, Neurobiology, 94:4109-4112, Bhardwaj et al., Journal of Immunological Methods, 1995, 179:165-175.

Schenk et al., each teach as set forth above meeting the limitations of claim 1, 8 and 12. However, neither Schenk reference explicitly teaches selection of bacteriophage species fd or M13 as in claims 4 and 7, having the properties of the limitations of claims 2-3.

Devlin et al., teach selection of filamentous bacteriophage fd and M13 for preferred production and presentation of peptide antigen, see in particular p. 6, lines 19-26.

The limitations of claims 2-3 are intrinsic properties of bacteriophage fd and M13 as evidenced by Bhardwaj. The filamentous phages are single stranded DNA phages which infect male specific E. coli strains (a normal constituent of host bacterial microflora), see in particular Bhardwaj et al., Journal of Immunological Methods, 1995, 179:165-175, p. 165, column 2.

Schenk et al. also do not teach the limitation of claims 6, 10 and 11 where the epitopes of Beta-amyloid are displayed via coat glycoprotein VIII on said bacteriophage.

Bhardwaj et al., (Journal of Immunological Methods, 1995, 179:165-175) teach selection of fd or M13 fusion constructs. Bhardwaj notes the advantages of VIIIp (glycoprotein) fusions for production and expression of foreign peptide epitope constructs in analyzing binding interactions interactions and the further benefit of the availability of specific monoclonal antibodies to either the minor (gIIIp) and major (gVIIIp) coat proteins of phage for analysis of expression, see in particular abstract and experimentation throughout.

Willis et al further teach that the genome of bacteriophage fd has been engineered to permit construction of hybrid virus particles in which the wild-type major coat protein (gpVIII) subunits were interspersed with coat proteins displaying one or other of two foreign peptides (fdMAL1, sequence NANPNANPNANP or fdMAL2, sequence NDDSYIPSAEKI) in the exposed N-terminal segments [Greenwood et al., J.

Mol. Biol. 220 (1991) 821-827]. These sequences represent major antigenic determinants of the circumsporozoite protein of the malaria parasite, *Plasmodium falciparum*. The peptide epitopes in the hybrid bacteriophages were found to be strongly immunogenic in four different strains of mice without the use of external adjuvants, and the antibodies (Ab) were highly specific to the individual epitopes in ELISA assays. When tested in nude (nu/nu) and heterozygote (nu +/-) BALB/c mice, the immune response was found to be T-cell dependent and to undergo class-switching from IgM to IgG. Proliferation assays of T-cells taken from lymph nodes of BALB/c mice injected with bacteriophage particles in the presence or absence of Freund's complete adjuvant indicated no difference in the immune response. This way of generating Ab against peptide epitopes is simpler and much less expensive than the conventional method of peptide synthesis and coupling to a carrier protein for injection. The specificity of the immune response, the ability to recruit helper T-cells and the lack of need for external adjuvants suggest that it will also be an inexpensive and simple route to the production of effective vaccines, see in particular abstract.

The artisan would have been motivated to select gpVIII constructs of either phage fd or phage M13 for expression of the Schenk epitopes of Abeta noted to comprise the elements of SEQ ID NO:1, evidenced to stimulate the appropriate immune response for inhibiting aggregation or providing for disaggregation of amyloid plaques. One of skill would be motivated to use phage expression as motivated by Schenk and further motivated by Bhardwaj and Willis to select gpVIII constructs for the recognized advantages of such vector constructs in providing expression, ease in analysis and

stimulation of an immune response in the host. One of skill in the art would have expected success using such presentation of antigen given the art recognized evidence for the suitability of the relevant epitopes for expression (Schenk), the high skill in the art of such expression using bacteriophage constructs (Bhardwaj, Willis and Devlin) and the advantages of gpVIII constructs in producing an immunogenic response and for ease of analysis of expression (Bhardwaj and Willis).

With respect to claim 5, the Examiner notes that the claim limitation is directed to steps to achieve administration such that a titer of antibodies is produced above 1:50,000. Schenk notes analysis of peptide expression such that titers are produced (so as to be effective in treatment and inhibition of aggregation of beta amyloid) such that the titers are directed to about 1:87,000 as determined by ELISA. The dosage is noted to be suitably tested and determined whether via peptide administration or via phage vector. In particular, Schenk teaches analysis of an effective amount via measurement of antibody titers and notes amounts inclusive of 1.0 μ g or 10 μ g or 50 μ g or 100 μ g (pg 3, line 25-7) effective to elicit antibodies and to effect removal or inhibition of plaque production/deposition. Schenk teaches mice, which developed a high antibody titer, including titers greater than 1/10,000 (see Figure 1). The highest antibody titers were generated with Freund's adjuvant with a peak GMT of about 87,000 as determined by ELISA. Schenk discloses if monitoring indicates a reduction of the immune response over time, the patient can be given one or more further doses of the agent (pg 4). Schenk discloses administration under conditions to generate a beneficial immune response (pg 2, line 35). Schenk et al. also teach the agent administered may be

displayed via nucleic acid production for presentation within the host and expressed upon the surface of a virus or bacteria, specifically via phage (bacteriophage) display methods (pg 3, line 28; pg 16, and 17, line 10-11) with reference noted therein to the art recognized teachings of Devlin, WO91/18980 as in claim 1 and that peptide libraries can be generated by phage display methods (pg 16-7). Accordingly, the artisan is motivated to provide phage dosage to mice such that at time point 30 days or less the titer is at least 1:50,00 as determined by ELISA. This corresponds to one of the most effective Schenk treatment regimes with AN1792 and 1528. In particular, "Mice were bled four to seven days following each immunization starting after the second immunization, for a total of five bleeds. Antibody titers were measured as A.beta.1-42-binding antibody using a sandwich ELISA with plastic multi-well plates coated with A.beta.1-42. As shown in FIG. 13, peak antibody titers were elicited following the fourth dose for those four vaccines which elicited the highest titers of AN1792-specific antibodies: AN1792 (peak GMT: 94,647), AN1528 (peak GMT: 88,231), A.beta.1-12 conjugate (peak GMT: 47,216) and rodent A.beta.1-42 (peak GMT: 10,766)."

Accordingly, the reference teachings render the invention obvious to the artisan.

15. Claims 1-4, 6-8, and 10 are rejected under 35 U.S.C. 103(a) as being unpatentable over Frenkel et al., Journal of Neuroimmunology, 1 August 1998, 88:85-90 (IDS reference AU; 7-15-03) in view of Bhardwaj et al., Journal of Immunological Methods, 1995, 179:165-175 and as evidenced by Winter et al., Ann Rev Immunol, 1994, 12:433-455.

Frenkel et al. (1998) teach anti-aggregating antibodies that bind to an epitope located as a continuous sequence at the beginning of the N-terminal of β -AP, and contemplates use of the peptide to control aggregation of β -AP in vivo (pg 86 and Results). The reference teaches (pg 85 and 86) epitope libraries in which different peptides are expressed on the surface of filamentous phage, including the peptide EFRH (Table 1) as in claim 8, which inhibits binding of monoclonal antibodies to β -amyloid peptide (claim 1). The reference teaches infecting *E. coli* K91 cells; and thus, the reference meets the limitations of a bacteriophage that propagates in bacterial flora in said recipient and in *E. coli* (claims 2-3).

Frenkel et al. do not teach use of the antigen displayed on the surface of a bacteriophage fd (claim 4) or M13 (claim 7) nor does it teach the epitope is displayed via coat glycoprotein VIII (claim 6 and 10).

Bhardwaj et al., (Journal of Immunological Methods, 1995, 179:165-175) teach fd or M13 and production of antibodies which react with the minor (gIIIp) and major (gVIIIp) coat proteins of phage M13. Bhardwaj also teaches constructs using these peptides for expression of foreign proteins in phage display and the advantages of such constructs in evaluating expression via monoclonal antibodies to the fused protein. Further, Winters et al. (1994) discloses selecting human antibodies of desired specificity.

It would have been obvious to one of ordinary skill in the art to use the teachings of Frenkel et al. and to use the bacteriophages fd or M13 disclosed by Bhardwaj et al to express the β -amyloid epitopes for the purpose of presenting the antigen to the immune system within the host for production of antibodies. The person of ordinary skill in the

art further would have been motivated to use the filamentous bacteriophage taught by Bhardwaj et al., that expresses foreign peptides or proteins displayed via coat glycoprotein VIII because Bhardwaj et al. teaches the advantages of their vector system in detection of foreign proteins through the use of the isolated monoclonal antibodies directed to glycoprotein VIII, and would have expected success because both Bhardwaj et al. and Frenkel et al., teach the use of the phage as a vector for expressing foreign peptide. Frenkel specifically motivates the artisan to substitute the A β peptide as the foreign peptide for the purpose of stimulating antibody production for detection and development of compounds to control aggregation of β AP in vivo, specifically for eliciting antibodies that inhibit aggregation and or cause disaggregation.

Conclusion

16. No claims are allowed.

17. Any inquiry of a general nature or relating to the status of this general application should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Papers relating to this application may be submitted to Technology Center 1600, Group 1640 by facsimile transmission. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). Should applicant wish to FAX a response, the current FAX number for Group 1600 is (703) 872-9306.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sharon L. Turner, Ph.D. whose telephone number is (571) 272-0894. The examiner can normally be reached on Monday-Thursday from 7:00 AM to 5:00 PM. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Janet Andres can be reached at (571) 272-0867.

Sharon L. Turner, Ph.D.
December 20, 2005


SHARON TURNER, PH.D.
PRIMARY EXAMINER
12-20-05